

Anti-Fascin (Ser-39), Phosphospecific Antibody

Catalog # AN1792

Product Information

Application	WB, IHC
Primary Accession	Q16658
Host	Mouse
Clonality	Mouse Monoclonal
Isotype	IgG1
Clone Names	M315
Calculated MW	54530

Additional Information

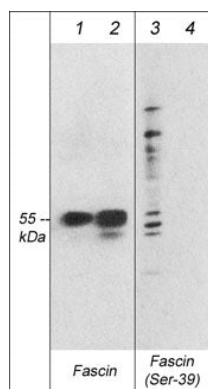
Gene ID	6624
Other Names	p55
Target/Specificity	Fascin is an actin filament bundling protein localized to lamellipodia and filopodia where it has important roles in cell motility. Regulation of fascin occurs through PKC-mediated phosphorylation of Ser-39 in the F-actin binding site. Cell permeant peptides that block PKC phosphorylation of Ser-39 increase cell migration, while peptides that block fascin binding to F-actin alter lamellipodial morphology and cause aberrant cell motility. Studies using RNA interference of fascin show that fibroblasts have reduced number and abnormal morphology of filopodia, while Ser-39 phosphorylation status may determine filopodial frequency. In <i>Drosophila</i> neurons, fascin deficiency causes alterations in actin filaments and leads to abnormal morphology of developing neurons. Thus, fascin is a critical element of actin-based motility in various cell types.
Dilution	WB~~1:1000 IHC~~1:100~500
Storage	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Anti-Fascin (Ser-39), Phosphospecific Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
Shipping	Blue Ice

Background

Fascin is an actin filament bundling protein localized to lamellipodia and filopodia where it has important roles in cell motility. Regulation of fascin occurs through PKC-mediated phosphorylation of Ser-39 in the F-actin binding site. Cell permeant peptides that block PKC phosphorylation of Ser-39 increase cell migration, while peptides that block fascin binding to F-actin alter lamellipodial morphology and cause aberrant cell motility. Studies using RNA interference of fascin show that fibroblasts have reduced number

and abnormal morphology of filopodia, while Ser-39 phosphorylation status may determine filopodial frequency. In *Drosophila* neurons, fascin deficiency causes alterations in actin filaments and leads to abnormal morphology of developing neurons. Thus, fascin is a critical element of actin-based motility in various cell types.

Images



Western blot analysis of mouse C2C12 cells treated with Calyculin A (100 nM) for 30 min (lanes 1 & 3) before treatment with lambda phosphatase (lanes 2 & 4). The blots were probed with anti-Fascin (clone 55K2) (lanes 1 & 2) and anti-Fascin (Ser-39) mouse monoclonal (lanes 3 & 4).

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.